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Is health wealth? Results of a panel data analysis

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The objective of this paper is to determine whether health (measured by life expectancy at birth) contributes to economic growth and the functional form in which it influences per capita income. This links our study to the debate between Neo-classical and endogenous growth theorists on whether investment in human capital can sustain growth indefinitely. Data on 216 countries for the period 1980-2009 has been obtained from World Development Indicators dataset. This enables us to focus on a period characterized by globalization and demographic changes manifested in the form of population graying. Our findings confirm the importance of investment in human capital. But, in contrast to conclusions of endogenous growth models, we find evidence that benefit from increasing longevity tapers off. We conclude by pointing out that it is necessary to extend this study further by incorporating other dimensions of health that are not captured by life expectancy.

1. INTRODUCTION

Economic growth is the increasing capacity of the economy to satisfy the wants of the society through provisioning of goods and services. It is enabled by the increase in productivity, which lowers the inputs required to produce a given amount of output.

The modern conception of economic growth began with the Physiocrats, who stressed on increasing productivity of the economy through capital formation.¹ Subsequently, the Classical economists, notably Smith (1776) and Ricardo (1817), argued that industrial growth fueled by the expansion of domestic and international markets was the driving force behind economic growth. The focus on productivity continued in subsequent developments. The Harrod-Domar model (Harrod, 1939; Domar, 1946) argued that economic growth depends on policies to increase investment, by increasing saving, and using that investment more efficiently through

¹ Quesnay's *Tableau économique* (1759) and Turgot's *Reflections on the formation and distribution of wealth* (1776) may be cited in this context (see Meek, 1962).

technological advances. The Harrod-Domar model was extended by the neo-classical economists (Solow, 1956; Swan, 1956) by including a new term, productivity growth. A major limitation of the Neo-classical growth models was their failure to explain the source of technological progress. This was a major failing, given that empirical studies attempting to explain economic growth in terms of labour and capital productivity found a strong residual effect. This shifted attention to other sources of growth, notably technological progress and human capital, and led to the development of endogenous growth models (also called AK models). These models depart from the neo-Classical models by arguing that technological progress is not exogenous, but is determined by socio-economic factors. In particular, investment in human capital, innovation and knowledge are significant contributors to economic growth. Further, two key components of human capital are the extent to which the labour force is educated, and the level of its health (Barro and Sal-i-Martin, 2004). This led to the formulation of theoretical models incorporating health as a determinant of economic growth (Barro, 1996; Grossman, 1972; Mankiw et al. 1992). Simultaneously, on the empirical front, studies like the 1993 World Development Report (World Bank, 1993) highlighted the critical role of health in growth and development.

Now, a major difference in the conclusions of the Neo-Classical and endogenous growth models were in the nature of growth. In particular, would investment in capital — particularly in human capital — sustain economic growth, as suggested by the AK models? Or, as the Solow-Swan models argued, did growth have its limits — returns to investment in human capital taper down to a steady state rate of growth? This question motivates the present study. Our objective is to see how the sweeping economic changes that are taking place since the 1980s (structural changes in economies leading to greater role for market forces and integration with world markets), coupled with the onset of a new demographic transition (in the form of increasing graying of the population) has shaped the health-wealth relationship.

The structure of the paper is as follows: In section 2 we briefly review the theoretical and empirical literature on the health-wealth relationship. This is followed by a description of the database and methodology in Section 3. Findings are presented and discussed in Section 4, followed by a concluding section summarizing our findings and indicating directions for future research.

2. HOW HEALTH AFFECTS PER CAPITA INCOME

In order to explain the relationship between health and economic growth, it is necessary to understand the concept of health in a broad sense. Health is not only the absence of illnesses; it is also the ability of people to develop to their full potential during their entire lives. In that sense, health is an asset individuals possess, having both an intrinsic value (being healthy is a very important source of well-being) as well as an instrumental value. This section focuses on the latter aspect.

2.1 Theoretical models

The importance of health was first emphasized by Mushkin (1962), who pointed out that health constitutes an important form of investment *distinct from other forms of human capital formation* like education. This idea was taken up and extended by Grossman (1972). Grossman's model views each individual as both a producer and a consumer of health. Health is treated as a stock which degrades over time in the absence of "investments" in health, so that health is viewed as a sort of capital.

In 1992, Mankiw et al. extended the Solow growth model by adding human capital, specifying that this variable has a significant impact on economic growth. Later, other authors developed models that included human capital, specifically health capital (Fogel, 1994; Barro and Sala-i-Martin, 1995; Barro, 1996). Following a Ramsey scheme, Barro (1996) developed a growth model including physical capital inputs, level of education, health capital and the quantity of hours worked. From first order conditions, he found that an increase in health indicators raised the incentives to invest in education, while an increase in health capital lowers the rate of depreciation of health. However, investment in health was also stated to be subject to diminishing marginal returns.

By and large, however, the first generation endogenous growth models focused on education, rather than on health. Initial works failed to develop a theoretically solid structure integrating health and economic growth (Gallego, 2000). This may be partly due to the lack of interaction between contributions in the field of health and in the field of economic growth, and partly due

to the difficulties in disentangling the effects of schooling and health on productivity, creating a bias towards the former. However, as pointed out by Aguayo -Rico et al. (2005), the impact of health is distinct from that of education in the sense that it varies through the entire course of life and is determined by behavior at various stages of life cycle. In spite of the focus on education, the endogenous growth models were an improvement over Neo-Classical models (Solow, 1956; Swan, 1956) - which argued that health affects only the *level* of GDP in the long run, and not its *growth* – by predicting that health has a permanent impact on the growth rate.

Second generation endogenous growth models,² or the “Schumpeterian” growth theory, developed recently (Aghion and Howitt, 1998; Howitt, 2000, 2005; Howitt and Mayer-Foulkes, 2005) identifies six different channels (productive efficiency, life expectancy, learning capacity, creativity, coping skills and inequality) through which an improvement in health of a country’s population will have an impact on its long-run growth performance. With the exception of life expectancy, these effects all work in the same direction. Specifically, they will raise the productivity and per-capita GDP of a country (both relative to the world technology leaders) that is sufficiently well off to be growing at the same rate as the world technology leaders, they will raise the growth rate of per-capita GDP in a country whose growth rate is below that of the technology leaders, and they will allow some countries finally to stabilize the relative gap in living standards that separates them from the technology leaders.

Increase in the life expectancy, on the other hand, reduces the skill-adjusted death rate and hence raises the skill per effective worker, increases steady-state capital stock per effective worker and also raises the country’s relative productivity. Subsequently, the effect will be to raise growth through two channels - the direct effect of raising income with which to finance technological investments and the indirect effect that works through increased capital accumulation which also raises income with which to finance technological investments which might overcome the disadvantage of backwardness and push it to the first convergence group.

² The second generation growth models differ from neo-classical growth models by arguing that technological progress is endogenous and varies between countries. However, it also takes into account the process of international technology transfer and its obstacles – so that the rate of technological progress becomes linked to global conditions (and not solely local conditions, as assumed in the AK growth models).

Other influences of life expectancy on growth are through *savings* and *education*. By lengthening the time horizon over which the return to saving and to education can be earned, an increase in life expectancy is likely to raise savings rate and enrollment rate. Increase in saving rate in turn will increase productive efficiency by increasing steady state level of capital stock.

2.3 Empirical studies

There is now a large body of empirical research on the relationship between health and economic growth. Research conducted by Strauss and Thomas in 1998 and Schultz in 1999 found that better health has a positive effect on the learning abilities of children, and leads to better educational outcomes. This increases the efficiency of human capital formation by individuals and households. Bloom, Canning and Sevilla (2004) examined the influence of health on growth based on more than a dozen cross-country studies. All these studies, with a single exception, show that health has a positive and statistically significant effect on the rate of growth of GDP per capita.

Fogel (1994) showed that about one-third of the increase in income in Britain during the nineteenth and twentieth centuries could be attributed to improvements in health and nutrition. Mayer (2001a) concluded that improvements in adult survival were causally linked to improvements in growth performance in Brazil and Mexico; Weil (2001) found that health (indicated by average height and LEB) explained about 17% of the variation in income per capita across countries. Gyimah-Brempong and Wilson (2004) found that 22% and 30% of the growth rate of per capita income in sub-Saharan Africa and OECD countries, respectively, can be attributed to health.

2.4 Life expectancy and growth

Cross-country macroeconomic studies suggest that longevity positively affects growth. For example, an increase in life expectancy from 50 to 70 years (a 40% increase) would raise the growth rate by 1.4 percentage points per year (Barro, 1996). A 10% decrease in malaria is associated with an increased annual growth of 0.3% (Gallup and Sachs, 2000) and malnutrition causes a decrease in the annual GDP per capita growth worldwide of between 0.23 and 4.7% (Arcand, 2001). For Latin America and the Caribbean, health, measured as the probability of

surviving to the next age group, has a strong long-term relationship with growth (Mayer 2001a). Using life expectancy and mortality rates as health indicators for different age groups, an estimate of the direct relationship between health and growth in Mexico from 1970-1995 indicates that health is responsible for approximately one third of long-term economic growth (Mayer 2001b). There are also several microeconomic studies that find a direct impact of adult health on productivity and income though the correlation is weaker than that found in comparative or historical study findings at national or regional levels (Moock and Leslie, 1986; Glewwe and Jacoby, 1995; Glewwe et al., 2001; Paxson and Schady, 2004).

Barro (1997) and Barro and Sala-i-Martin (2004) used a panel dataset of countries, and incorporated life expectancy at birth (LEB) as a proxy for health along with years of educational attainment and other factors that could potentially influence the growth of real income per capita. Their results indicate that the log of LEB has a positive and statistically significant effect on growth rate with a coefficient of 0.042, which implies an annual rate of increase of per capita real GDP of 4.2%. Bloom, Canning and Sevilla (2004) review several studies that include health as an explanatory variable in growth equations, in addition to presenting new results, based on a cross-national panel dataset for countries. They use a production function model of economic growth with a measure for human capital which takes account of the indicators of health, education and labour market experience. They report a positive and statistically significant effect of health on economic growth. Their empirical findings reveal that an increase of one year in LEB raises the growth rate of GDP by 4%.

Azomahou (2008) attempts a nonparametric inference of the relationship between life expectancy and economic growth. He considered data on 18 countries over the period 1820-2005. He showed that the relationship was convex for low values of life expectancy, but became concave for larger values of life expectancy. Among other studies indicating non-linearity in the relationship between life expectancy and growth is that of Faruquee and Mühleisen (1997). This study finds a hump-shaped relationship between longevity and economic growth. Another study of 47 countries by Cervellati and Sunde (2011) observes a V-shaped relationship between wealth and life expectancy.

A recent study by Hansen (2012) explores the non-linearity dimension using 10-yearly observations from 1940 to 1980 with data from Acemoglu and Johnson (2007). Hansen finds a non-monotonic relationship between life expectancy and GDP per capita. Specifically at early stages of development, the effect of health is to increase the size of the population which may have a negative impact on wealth. But at higher stages of development, health improvements induce human capital skills which may in turn actually lead to a lower population size thus encouraging an increase in GDP per capita.

2.5 Introducing endogeneity

While there is compelling evidence that health contributes significantly to economic growth, there is also voluminous literature that focuses on causality in the reverse direction - from income to health. Pritchett and Summers (1996) estimate the effect of income on health, measured by infant and child mortality as well as life expectancy. The existence of an impact of health on economic growth with similar magnitudes has been verified for different time periods and countries, including Latin America and Mexico (Preston 1975, Deaton 2001). In response to this, there has been some empirical works that have attempted to incorporate the endogeneity embedded in the relationship between growth and health.

Aguayo-Rico et al. (2005) tried to determine the effect of variable like capital, labor, schooling, lifestyles, environment, health services and the total health index on absolute values of growth per capita. They also wanted to see which variable had the biggest impact on economic growth. For this they collected data for 52 countries on various determinants for the periods 1970-1980 and 1980-1990. It was observed that all variables were significant at the five percent level of significance and had the expected signs. Re-estimating the model using growth rates in per capita levels, rather than in absolute levels and including the total health index as the health variable, it was found that all variables are significant at the one percent level except schooling. When the model was re-estimated using GLS technique, they found that the coefficients were more robust and efficient. Further, all variables were significant at one percent level of significance, showing that generalized least squares method performed as a better estimation technique.

On the other hand, when the model was estimated using life expectancy as the health variable, following the specification of Bloom et al. (2004),³ it was seen that life expectancy was statistically insignificant. This indicated that life expectancy might not be a good representation of health capital.

Another important work in this line is by Acemoglu and Johnson (2007). Based on estimates of mortality by disease before the 1940s from the League of Nations and national public health sources, the authors constructed an instrument for changes in life expectancy (referred to as predicted mortality) which is based on the pre-intervention distribution of mortality from various diseases around the world and dates of global interventions. Acemoglu and Johnson concluded that predicted mortality has a large and robust effect on changes in life expectancy starting in 1940, but no subsequent effect on changes in life expectancy. In contrast, life expectancy has a much smaller effect on total GDP, both initially and over a 40-year horizon. Consequently, these results confirm that global efforts to reduce poor health conditions in less developed countries can be highly effective, but at the same time it is also doubtful whether unfavorable health conditions are the root cause of the poverty of some nations.

3. RESEARCH OBJECTIVE AND METHODOLOGY

3.1 Research question

This paper examines the role of life expectancy at birth in determining per capita income of an economy. Our hypothesis is that increasing longevity affects per capita income positively, though not necessarily in a linear relationship. Further, it has an independent impact even after incorporating the effects of increasing savings, educational attainments, fertility rate and public spending on health. We will also try to examine whether investing in health can sustain growth indefinitely, or whether there are diminishing returns to such investment. This will link our research question to the theoretical literature on growth theory – in particular, whether growth has its limits, or it may be sustained indefinitely.

³ This was done in order to observe the statistical impacts made by the different health measures on economic growth.

Now, there has already been a vast body of work on the health-wealth nexus. However, such studies generally have employed data up to 1980, or at most up to 1990. Given the existence of several studies showing that globalization has increased longevity (Owen and Wu, 2007; Stroup, 2007; Ovaska and Takashima, 2006; Bergh and Nilsson, 2010), an interesting exercise would be to study the link between life expectancy and wealth in the post-1980 period, when the seeds of the sweeping globalization and liberalization of the 1990s were being sowed through World Bank funded Structural Adjustment Programmes in developing countries. Moreover, the 1990s and 2000s are characterized by a new demographic process, in the form of rapid increase in population aged 60 years and above (UN, 2005). This has important implications for social security policies as the increase in dependency ratio and decrease in their support base calls for appropriate policy response in the form of increased resource allocation on health, pension and social security of the aged (Auerbach et al., 1989; Heller, 1989; Cutler et al., 1990; Halter and Hemming, 1987; Masson and Tryon, 1990; Hu, 1995; Dahan and Tsiddon, 1998).

3.2 Database

The study uses data on life expectancy at birth (LE) and gross national income per capita (at purchasing power parity, measured in current international US dollars — GNI) from the World Development Indicators dataset from the World Bank site (www.data.worldbank.org/indicator, accessed in December 2011) for the years 1980-2009. The WDI indicators included data for 216 countries.

The use of life expectancy at birth as a proxy for health requires some justification. This is in line with empirical studies in this field. Most of the studies on the linkage between growth and health reviewed in section 2 take longevity as a proxy for health, and do not consider the other dimensions of health. If the true value of health has to be assessed, however, health should be measured in all its multiple dimensions - mortality, morbidity, disability and discomfort. Life expectancy takes into account only mortality, but it is not perfectly correlated with the rest of the health dimensions (Evans et al. 1994). Moreover, life expectancy reveals only the lifetime of the stock of human capital, saying nothing about the time in the labor force of this capital or the problems caused by population aging. This is a problem because, even though there is a strong

connection between health, productivity and economic growth, health capital depreciates over time (Grossman 1972) and at one point the relationship stops being binding.

However, longevity does emphasize the duration of health status and places implicit importance on a persons' well-being (Jacobs and Rapaport, 2002). Further, it is a variable that is easy to conceptualize and to measure.⁴ Hence, despite its limitations, health continues to be captured by life expectancy in empirical studies. This study tries to balance both view points. While we accept the limitations of life expectancy in capturing multiple dimensions of health, we feel that this is a variable *whose impact on wealth is sufficiently strong and interesting enough to be studied independent of other dimensions of health*. Hence we will focus explicitly on the relationship between longevity and wealth – deliberately ignoring the other (equally important) dimensions of health.

3.3 Methodology

The analysis uses both bivariate and multivariate methods. We first conduct a graphical analysis of log of gross national income per capita (LGNI) against log of life expectancy at birth (LLET) for all the countries over the period 1980-2009. The graphical analysis is then repeated for five-yearly intervals – for the years 1980, 1985, 1990, 1995, 2000, 2005 and 2009 – based on the reasonable assumption that the relationship between LGNI and LLET will not change significantly over such a short period.

The multivariate method uses panel data models. We first estimate the reduced form model of LGNI on LLET.⁵ A non-linear specification is estimated. This specification allows us to address increasing concerns related to population aging caused by rising longevity in many countries, with adverse impact on the macroeconomic and social front. In the second step, we introduce endogeneity by incorporating lagged values of LGNI to estimate the Arellano-Bond dynamic panel model. In the final step we extend the reduced form model by introducing possible

⁴ For instance, QALY-adjusted life expectancy appears to be a more relevant variable for our purpose as it captures quality of life dimension. Unfortunately, we have country-wise data on only one year.

⁵ Economic growth is determined by economic factors, socio-cultural processes and human capital. Structural form enables us to capture the impact of all these factors on growth. However, reduced form models is an important preliminary step in analyzing macroeconomic relationships as it enables us to focus directly on the relationship between health and economic growth, assuming other factors to be constant.

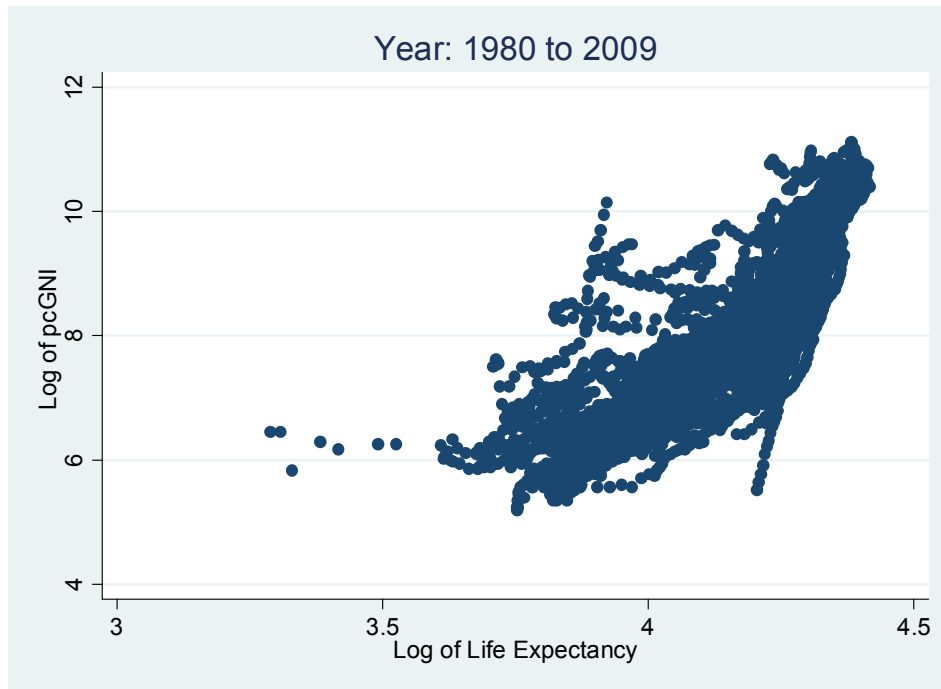
channels through which LLET affects LGNI. Given that there may be inter-relationship between LLET and LGNI the complete form model is estimated using the Instrumental Variable method.

4. FINDINGS

4.1 Bivariate analysis

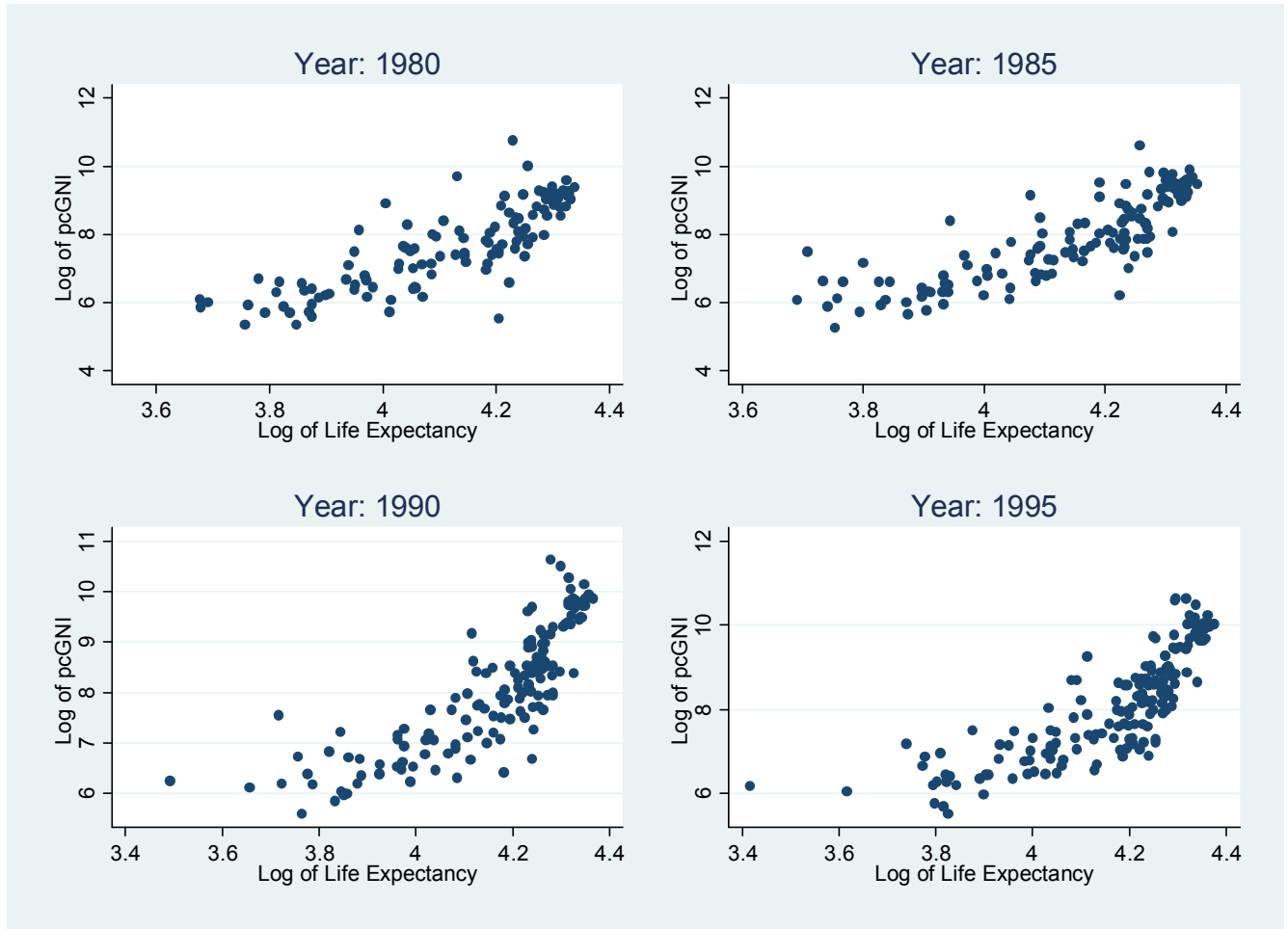
We plot LGNI and LLET for all the years (1980-2009) across all the 216 countries. The resultant scatter is given in Figure 1. We see that the relationship between log of life expectancy and log of per capita income is positive but non-linear. Specifically, it is convex from below.

Figure 1: Scatter plot of (log of) longevity and (log of) per capita GNI – 1980-2009



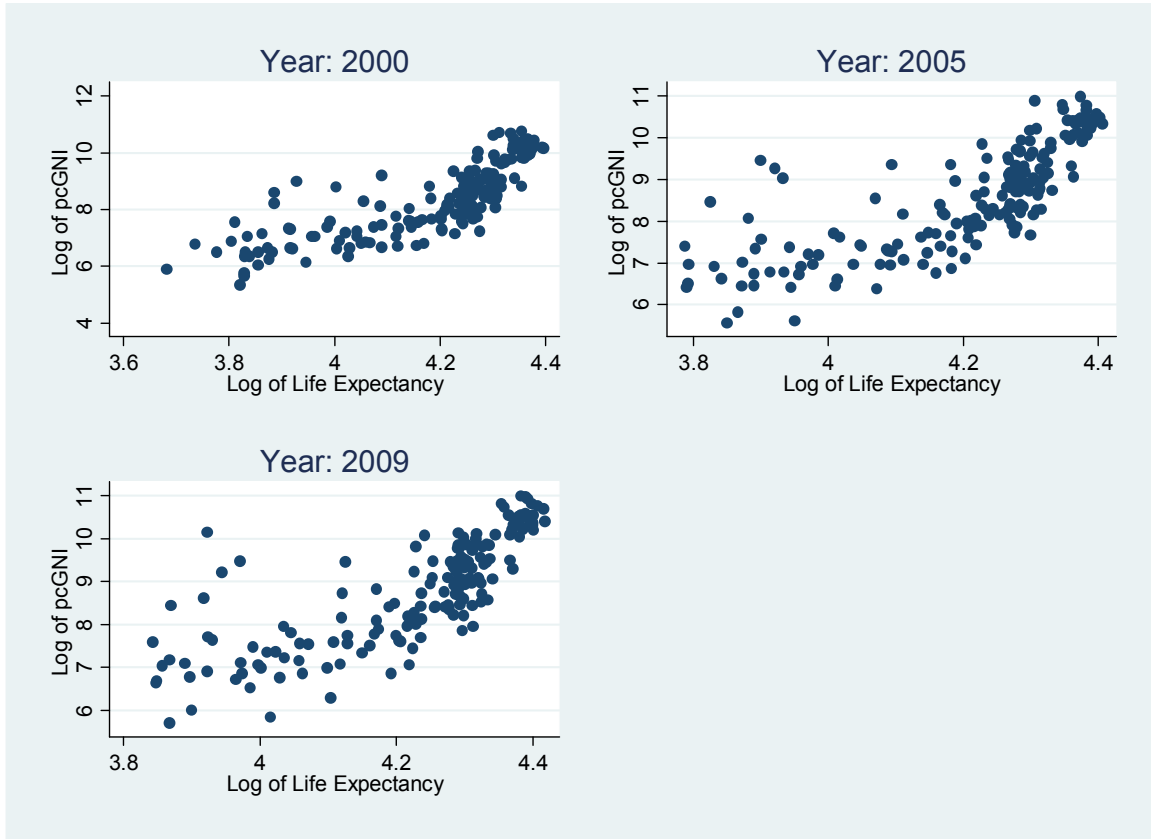
The following graphs (Figure 2) give the scatter for all countries but for particular years like 1980, 1985, 1990 and 1995. We see that the relationship is positive and weakly convex. The fit is close in all cases.

Figure 2: Scatter plot of (log of) longevity and (log of) per capita GNI – 1980, 1985, 1990 and 1995



When we plot the graph for the years 2000, 2005 and 2009 (Figure 3), however, we obtain a different distribution. The fit is not as close as before in for countries with shorter average life spans. More important, the initial rising phase is followed by a period when per capita wealth remains steady, or even falls, with increases in longevity. In the third phase, we see a resumption of the positive link between life span and wealth.

Figure 3: Scatter plot of (log of) longevity and (log of) per capita GNI – 2000, 2005 and 2009



4.2 Reduced form

We first regress the equation:

$$\text{LGNI}_{it} = \alpha_1 \text{LLE}_{it} + \alpha_2 \text{LLE}_{it}^2 + \text{LLET}_{it}^3 + \delta_t + \eta_i + v_i \quad [1]$$

The first steps in panel data analysis is to test whether (individual) country-specific effects exist and (if they do exist) the nature of such effects. The hypothesis that there are no individual specific effects may be tested using the Breusch-Pagan test (Breusch and Pagan, 1979). The value of the χ^2 statistic is 43680.56 ($p=0.00$), which indicates that the null hypothesis may be rejected at 1% level. Given that individual country-specific effects exist, the second question is whether such effects are correlated with life expectancy (fixed effects model) or that they are not (random effects model). The hypothesis of random effects is tested using the standard Hausman test (Hausman, 1978). The χ^2 statistic of 259.51 ($p=0.00$) rejects the hypothesis that life expectancy and country effects are uncorrelated, indicating that the fixed effects model (FEM) is appropriate. Results of the FEM are reported in Table 1.

Table 1: Results of Fixed Effects Model

Variables	All Countries		Reduced Sample		Lagged Model	
	β	t-ratios	β	t-ratios	β	t-ratios
LLET	247.67	13.02***	434.77	10.69***	399.39	10.38***
LLET ²	-65.82	-13.60***	-111.95	-11.10***	-102.08	-10.68***
LLET ³	5.82	14.19***	9.61	11.54***	8.70	11.00***
Time dummies	Present		Present		Present	
Constant	-302.68	-12.16	-555.28	-10.15***	-512.74	-9.94***
F	451.03	0.00***	457.51	0.00***	460.11	0.00***
R ² (overall)	0.56		0.57		0.52	
N	4715		4674		4818	

Note: *** $p < 0.01$.

Model with fifth lag has been reported.

We see that all the coefficients are statistically significant individually, as is the overall model. Given the signs of the coefficients, (log of) wealth appears to have an N-shaped relation with (log of) life span. The two turning points are estimated to be 36.81 years and 51.09 years. This means that, till the age of 37 years, there is a positive relationship between log of per capita GNI and log of life expectancy. The same relationship holds for age above 51 years. But for age lying between 37 and 51 years, the relationship is negative. The N-shaped curve is somewhat surprising and needs to be examined more closely to rule out the possibility that this is a statistical artifact.

Turning points of the cubic model are 36.81 and 51.09 years. Since the former point is quite low, it may be worthwhile to check how many countries actually have LE values below 36.81. It appears that only Timor-Lestis and Rwanda have values of LE below the lower turning point. Hence the above models are re-estimated dropping these two countries. Identical results are obtained. The cubic form (with t-ratios in parentheses) has turning points at 45.48 and 52.04 years, with 300 observations in the dataset below the lower turning point.

Another possibility is to incorporate lagged values of LLE into our model. The logic behind this is that investment in health will affect growth, not immediately, but after a time lag. Incorporating lags involves choosing the appropriate length of the lag. We have estimated lags running from one to five, getting an N-shape in each case. Further, the explanatory power of the models (R^2) gets reduced as we increase the lag. Thus, taking lags does not really add much to the picture. Results of the model with five lags are stated in Table 1.

4.3 Explaining shape of wealth-health curve

Hansen (2012) had explained the U-shaped nature of the relationship between life expectancy and wealth observed for the period 1940-1980 in terms of demographic transition and its links with the Malthusian trap. In initial stages of growth, as the only impact of rising longevity is to increase population size the relationship between wealth and life expectancy is negative. Subsequently, however, “health improvements may induce human capital skills and the so-called Malthusian population-link may be broken so that health improvements actually lead to a lower population size” (Hansen, 2012: 175-6). From this stage on, the wealth-longevity curve becomes positively sloped.

By the 1980s, however, the demographic transition had ended in most countries so that most countries may be expected to have crossed the turning point and be on the positively sloped section of the wealth-longevity curve. Moreover, the changing techno-economic context - the process of economic liberalization occurring in many countries, along with associated changes in economic structure, establishment of greater links between countries and rapid increase in technological progress (and its transfer) – may be expected to have modified the wealth-longevity link. However, econometric analysis indicates that, after the 1980s, the life expectancy-per capita income curve is N-shaped. While the positively sloped sections of the curve is expectedly in tune with Hansen’s findings and need no explanation, the downward sloping section of the wealth-longevity curve deserves attention.

In Figure 3, we had shown scatter plots for the years 2000, 2005 and 2009.⁶ These plots also reveal an N-shaped relation between the two variables of interest. We then identified countries

⁶ Scatter plots for earlier years revealed a positively sloped curve.

lying in the negatively section of the wealth-longevity curve. It was observed that countries like Bangladesh, Bhutan, Bolivia, Cambodia, Ghana, Guyana, India, Indonesia, Mongolia, Myanmar, Namibia, Pakistan, Sudan, Turkmenistan and Yemen fall in this (negatively sloped) section of the curve for *all* the three years. One possibility is that these countries have poorly developed health sectors, which have failed to make any contribution to the growth process. For instance, in a recent article, Sen and Dreze (2011) have pointed out that, despite India's success in economic growth, her health outcomes are among the worst in the world.

To verify this explanation we have constructed factor scores based on data on some health indicators that are used by policy makers. These variables are children *not* immunized for measles and DPT, infant mortality rate, child mortality rate and maternal mortality rates for 2010.⁷ Since data was missing for some countries, we were able to estimate scores for only 172 countries. We found that 26 out of the 29 countries lying in the negatively sloped portion of the wealth-longevity curve were among the 63 countries with lowest factor scores (that is with poorest health outcomes). Thus, there is some evidence that the falling section of the curve reflects the fact that growth, in some developing countries, has not been fueled by human development but has occurred mainly due to techno-economic processes. In these countries, the neglect of the social sector in the bid to accelerate economic growth may have distorted the otherwise positive relationship between health and per capita income levels.

4.4 Grouping countries

Finally, we have estimated regression [1] for each of the groups of countries, classified by their per capita income levels in 2009, and examine the robustness of our initial results for each group. We classify the sample into four groups using the World Bank classification. The four groups are:

- a) High income: With per capita income above \$12276
- b) Upper middle income: With per capita income between \$3,976 - \$12,275
- c) Lower middle income: With per capita income between \$1,006 - \$3,975
- d) Low income: With per capita income \$1,005 or less

⁷ The Kaiser-Meyer-Olkin statistic was 0.771, Bartlett's Test statistic was 1274.32 ($p=0.00$); one eigenvalue with value of 3.98 explaining 79.61% of the variation was extracted.

Results are reported in Table 2.

It is interesting to note that the cubic form is persistently observed for all groups barring Lower middle income countries. For this group, the quadratic form

$$\text{LGNI} = 58.5635 - 27.4018 \text{ LLE}_{it} + 3.6007 \text{ LLE}_{it}^2$$

(-7.67) (8.07)

N: 1163; Groups: 46; F: 125.02

is found to be appropriate. This indicates a U-shaped relationship between LLET and LGNI. We should also note that the coefficients of the quadratic form in remaining three groups are insignificant at 10% level.

Table 2: Regression results for country groups

Variables	High income		Upper middle income		Lower middle income		Low income	
	β	t	β	t	β	t	β	t
LLE	843.0939	5.70***	294.7413	2.14***	56.2240	0.53	287.0430	1.92*
LLE ²	-202.4582	-5.68***	-73.6585	-2.16***	-17.2306	-0.65	-71.9598	1.89*
LLE ³	16.2065	5.66***	6.1541	2.19***	1.7282	0.79	6.0163	1.86*
Intercept	-1160.294		-385.8286		-53.2405		-374.8611	
N	1746		1304		1163		461	
Countries	66		50		46		19	
F	345.01		123.93		121.09		5.85	

Note:

All regressions include time fixed effects.

**** implies $p < 0.01$, ** means $p < 0.05$ and * means $p < 0.10$.*

4.5 Endogeneity

A parameter or variable is said to be endogenous when there is a correlation between the parameter or variable and the error term. Endogeneity can arise as a result of measurement error, autoregression with autocorrelated errors, simultaneity, omitted variables, and sample selection errors. Broadly, a loop of causality between the independent and dependent variables of a model leads to endogeneity. For instance, here the independent variable (life expectancy) itself depends on the dependent variable, per capita income (see Preston, 1975).

Another possible form of endogeneity – one that we shall consider here – is through the introduction of lagged value of the dependent variable. The purpose is to capture the momentum of growth, apart from the impact of health on growth. In such situations, when the independent variable and the error term are correlated, LSDV (Least Square with Dummy Variables) estimates of the regression coefficient may be biased. If other regressors are correlated with the lagged dependent variable, then their coefficients too may be biased. Even use of the REM will not solve this problem as the error component (u_i) enters every value of y_{it} , so that lagged dependent variable becomes dependent on the composite error process.

Anderson and Hsiao (1981) suggest that in this case we can take the first difference of the original equation and use lagged values of the dependent variable as instruments. The differencing method, however, has been criticized by Arellano and Bond (1991). They argue that this approach does not exploit all the information available in sample. Secondly, even though consistent estimators are obtained, the method fails to take all the potential orthogonality conditions into account. In this situation, they argue, Generalized Method of Moments (GMM) can yield more efficient estimators — referred to as the Arellano-Bond model.

Now, one problem with the Arellano-Bond model is that such models may have a large number of instruments (in our model we would have 438 instruments). In such cases, the Sargan test of over identifying restrictions loses its robustness. A possible option is to increase the lag. Even after we increased the number of lags and repeated the Sargan test, we found that the statistic was not robust. Another alternative is to estimate the LSDVC model, which also corrects for bias

due to the unbalanced nature of the panel (Bruno, 2005). The bootstrapped estimates of the LSDVC dynamic regression model are stated below:

$$\text{LGNI} = 0.98 \text{ LGNI}_{it-1} + 0.29 \text{ LLE}_{it}^2 - 0.05 \text{ LLE}_{it}^3 \quad (+ \text{Time dummies})$$

(109.61) (3.65) (-3.56)

Here, we see that all the coefficients, i.e. lagged LGNI, LLE_{it}^2 and LLE_{it}^3 are statistically significant, while LLET is dropped because of collinearity. Further, the coefficient of LLE_{it}^2 is positive whereas the coefficient of LLE_{it}^3 is negative. This means that health-wealth curve is an inverse U-shaped curve – increasing up to a point, before falling. This may be ascribed to the new demographic transition that is taking place in many countries – the increase in the proportion of elderly persons (over 60 years) in the total population.

The sharp dip in fertility rates in recent years, combined with falling mortality rates over the last four decades, is leading to an increase in the absolute and relative size of the elderly population in developing countries (Rajan *et al.*, 1999). Population projections indicate an increasing ‘greying’ of the world’s population, with the share of persons aged 60 years and above predicted to increase from 9.5% in 1995 to 30.5% in 2150 (United Nations, 2005). The growth rate of the elderly population varies between countries, but is expected to be high in Afro-Asian countries. In India, for instance, the share of aged has increased from 6.5% (1981) to 7.4% (2001), and is expected to constitute about a quarter of the population by 2075. Although some researchers argue that the increase in the elderly population merely comprises a change in dependency structure, with resources freed from a reduction in the share of the young being sufficient to take care of the elderly (Ball & Bethell, 1998), the issue is more complex, particularly in developing countries, and goes beyond looking after the special needs of the elderly with respect to health care, housing and financial insecurity (Nyce & Schieber, 2005). In these countries, as Palloni (2001) points out, ageing of the population is occurring in economic, social and political contexts that are often fragile and precarious. This implies that a considerable amount of resources will have to be diverted away from productive uses to creating a social security system for the aged. In the long run, therefore, increasing longevity may adversely affect growth so that the relationship between them becomes negative.

4.6 Incorporating channels of health-wealth interaction

Here, an increase in life expectancy affects growth through various channels (Acemoglu and Johnson, 2006; Hazan, 2011, Howitt, 2005; Zhang and Zhang, 2005). In this section, we introduce these factors - fertility rate (FRATE), gross savings (SAVING), primary completion rate (PCR), public spending on education (PSEDU), tertiary school enrollment⁸ (TEDU) — into our model. The data is extracted from the World Bank Development indicators used earlier.

Now one important problem with the model:

$$LGNI = F(LLET, SAVING, FRATE, PCR, TEDU, PSEDU, \text{Year Dummies}) \quad [2]$$

is that LLET is also determined by LGNI (Preston, 1975). This indicates the presence of endogeneity so that we have a simultaneous equation system as follows:

$$LGNI = F(LLET, *) \quad [3]$$

$$LLET = G(LGNI, **) \quad [4]$$

Such a system can be solved by introducing an instrument (a variable that affects LLET but not LGNI) in the equation $LLET = G(LGNI, **)$. We then estimate the equation $LLET = G(\text{INSTRUMENT}, **)$, and plug in the *predicted values* of LLET in the equation $LGNI = F(LLET, *)$. The problem with this method is that it is not very easy to identify a variable that affects LLET but not LGNI. In general, the better the instrument, the better is the specification of the model, and the better is the results. In our analysis, we have taken infant mortality rate, denoted by IMR, as the instrument variable.

In the first stage the instrument IMR has a coefficient -0.0038 with a z-value of -23.14 ($p=0.00$). This supports our proposition that this instrument significantly affects the life expectancy at birth. The negative sign is also expected because a high mortality rate at birth should also reduce average life span. In second stage, we use the predicted values of LLET obtained from first stage and run the regression (log of) GNI on predicted LLET and other regressors like savings, primary and secondary education, per capita high expenditure fertility rate and primary completion rate. This allows us to check whether LLET has an individual effect on GNI apart

⁸ The proportion of individuals in relevant age group enrolled in educational institutions (colleges, universities and polytechniques) offering post-secondary education.

from its effect through the other regressors. The second stage results obtained are reported in Table 3 for two models – Model 1 (represented by [2]) and Model 2 (Model 1, with slope dummies for countries classified by World Bank income criterion used earlier).⁹

Table 3: Results of IV regression

Variables	Model 1		Model 2	
	Coefficients	Z	Coefficients	Z
LLET	2.40	7.53***	2.12	4.04***
SAVING	0.01	7.00***	0.01	7.97***
PCR	-0.0004	-0.42	0.0001	0.09
TEDU	0.01	8.99***	0.003	3.46***
PSEDU	0.03	4.57***	0.03	3.88***
FRATE	0.01	0.79	0.10	5.67***
WBS1			-0.13	-0.25
WBS2				
WBS3			-1.48	-2.84***
WBS4			-2.52	-4.52***
CONSTANT	-2.35	-1.75*	1.09	0.86
N	766		766	
Wald χ^2	4.27e ⁶		4.29e ⁶	
ρ	0.99		0.99	
R ²	0.43		0.56	
F	127.44		72.50	

Note: *** $p > 0.01$, * $p > 0.10$

All regressions include time fixed effects.

The value of the Wald χ^2 indicates that the overall model is significant. We also find the coefficient of LLET to be quite high (2.40 and 2.12, respectively). This shows LLET does have an individual effect on GNI, apart from indirect effects through the other regressors.

⁹ We have run Hausman tests to identify Fixed Effect variant as appropriate variant of models.

The signs of all the variables are expected except for that of PCR (in both models) and FRATE (in first model). The sign of PCR in Model 1 is also unexpectedly negative. This may be because of multicollinearity — both PCR and FRATE are highly correlated with TEDU (0.60 and -0.70, respectively).

Now, one objective in taking slope dummies was to see how the effect of longevity changes as we move to high income groups. Results show that the coefficient of LLET for the four income groups are 2.12, 2.12, 0.64 and -0.40. In other words, the impact of increasing longevity declines if we consider higher income countries. Thus, investment to health experiences diminishing returns as growth occurs. This contradicts endogenous growth models and is in line with the earlier Swan-Solow Neo-Classical models.

5. CONCLUSION

In this study we have examined the relationship between life expectancy and per capita income using country-level panel data from 1980-2009. Results show that this relationship is non-linear. While the reduced form model indicates that the life expectancy-per capita income curve is N-shaped. The existence of a downward sloping section in the health-wealth curve is somewhat surprising and has been examined carefully. We find that the finding is robust even if we divide countries into income groups, or introduce lagged values of health. A possible reason for this phenomenon may be that some countries base their growth entirely on techno-economic forces – neglecting the social sector. The introduction of endogeneity, however, modifies the shape of the health-wealth curve. Results indicate that the curve may be inverse U-shaped. The latter implies that, beyond a point, increasing life expectancy may impose costs on society by increasing the proportion of elderly in population. Investment in social security (pension), and provisioning of housing and health services to such persons may divert resources away from productive resources, without any corresponding economic benefits.

Subsequently, we have estimated a complete form model to incorporate channels through which life expectancy may be expected to stimulate growth. This model retains the endogenous structure. Results of the instrumental variable model reveals that the hypothesized channels (savings, expenditure on health, fertility, expenditure on education, and enrolment in primary and

tertiary levels) through which life expectancy operate are all significant. Further, life expectancy continues to have an effect on per capita income independent of these channels. This strongly underlines the vital importance of investing in human capital (in this case, in the form of life expectancy) for economic growth. This matches with developments in the literature on growth models.

However, does investment in human capital sustain growth indefinitely? Or, does growth have its limits? The Neo-classical models of Swan and Solow had argued that growth declines asymptotically to the steady state rate of growth, given by the exogenous rate of technological progress. However, the endogenous growth models had showed that the limits to growth imposed by the exogenous rate of technological progress may be eliminated through investment in human capital (Lucas, 1988; Romer, 1990). Our results do not confirm the policy implication flowing from the endogenous growth model, viz. that investment in health will generate marginal benefits on a continuous basis. Rather, in keeping with the findings of the Neo-Classical models, we find that returns to investment in longevity will decline as growth occurs and will ultimately taper off. This highlights that aging will increasingly become an obstacle to the growth process over time. We suggest, therefore, that while health may still be wealth, investing in increasing longevity may not be so beneficial beyond a point.

However, we should also add an important rider. Equating longevity with health may not be appropriate as the former is a factor distinct from other health aspects and has negative consequences for growth. Exploring the relation between per capita income and other dimensions of health is important to before concluding that investment in health cannot sustain growth. This is a promising area for future research.

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